WHAT IS CLAIMED IS:

1. A method of treating a subject diagnosed as having a lysosomal storage disease comprising administering a gene therapy vector encoding a lysosomal hydrolase under the control of at least one tissue specific regulatory element and administering:

(a) an exogenously produced natural or recombinant lysosomal hydrolase;

(b) a small molecule capable of treating a lysosomal storage disease, or

(c) both (a) and (b),

such that the lysosomal storage disease is treated.

2. The method of claim 1, where the gene therapy vector encoding a lysosomal hydrolase under the control of a tissue specific regulatory element is administered before the exogenously produced natural or recombinant lysosomal hydrolase or the small molecule capable of treating a lysosomal storage disease.

3. The method of claim 1, where the tissue specific regulatory element is chosen from at least one of a tissue specific promoter and a tissue specific enhancer.

4. The method of claim 1, where administering the gene therapy vector encoding a lysosomal hydrolase induces immunological tolerance to the lysosomal hydrolase.

5. The method of claim 1, where administration of the gene therapy vector encoding a lysosomal hydrolase under the control of a tissue specific

promoter is followed by administration of an exogenously produced natural or recombinant lysosomal hydrolase.

6. The method of claim 5, where the amount of the exogenously produced natural or recombinant lysosomal hydrolase administered to the subject is less than the amount administered to treat a subject with a lysosomal storage disease that has not been administered a gene therapy vector encoding a lysosomal hydrolase or has been administered a gene therapy vector without a tissue specific promoter controlling expression of the lysosomal hydrolase.

7. The method of claim 1, where the lysosomal storage disease is Fabry disease.

8. The method of claim 7, where the treatment results in a decrease in GL-3 in the subject compared to the GL-3 level in the subject before treatment.

9. The method of claim 7, where the lysosomal hydrolase is α-galactosidase A.

10. The method of claim 1, where the lysosomal storage disease is Pompe disease.

11. The method of claim 10, where the treatment results in a decrease in glycogen in the subject compared to the glycogen level in the subject before treatment.

12. The method of claim 10, where the lysosomal hydrolase is α-glucosidase.

13. The method of claim 1, where the gene therapy vector is a viral vector.

14. The method of claim 11, where the viral vector is chosen from AAV1, AAV2, AAV5, AAV7 and AAV8.

15. The method of claim 1, where the tissue specific regulatory element is a liver specific promoter.

16. The method of claim 15, where the liver specific promoter is a human serum albumin promoter.

17. The method of claim 1, where tissue specific regulatory element is a tissue specific enhancer.

18. The method of claim 17, where the tissue specific enhancer is a human prothrombin enhancer.

19. The method of claim 1, where the small molecule capable of treating a lysosomal storage disease is chosen from deoxynojirimycin, N-propyldeoxynojirimycin, N-butyldeoxynojirimycin, N-butyldeoxygalactonojirimycin, N-pentlydeoxynojirimycin, N-heptyldeoxynojirimycin, N-pentanoyldeoxynojirimycin, N-(5-adamantane-1-ylmethoxy)pentyl)-deoxynojirimycin, N-(5-cholesteroxypentyl)-deoxynojirimycin, N-(4-adamantanemethanylcarboxy-1-oxo)-deoxynojirimycin, N-(4-adamantanylcarboxy-1-oxo)-deoxynojirimycin, N-(4-phenantrylcarboxy-1-oxo)-deoxynojirimycin, N-(4-cholesterylcarboxy-1-oxo)-deoxynojirimycin, or N-(4-b-cholestanylcarboxy-1-oxo)-deoxynojirimycin, D-threo-1-phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (P4), D-threo-4'-hydroxy-1-phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (4'-hydroxy-P4), D-threo-1-(3',4'-trimethylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (trimethylenedioxy-P4), D-threo-1-(3',4'-methylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (methylenedioxy-

P4) and D-threo-1-(3',4'-ethylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (ethylenedioxy-P4 or D-t-et-P4).

20. A method of treating a subject diagnosed as having Fabry disease comprising administering a gene therapy vector encoding α-galactosidase A under the control of a human albumin promoter and 2 copies of a human prothrombin enhancer and administering:

(a) an exogenously produced natural or recombinant α-galactosidase A;

(b) a small molecule capable of treating Fabry disease, or

(c) both (a) and (b),

such that the Fabry disease is treated.

21. The method of claim 20, where the gene therapy vector encoding α-galactosidase A under the control of a human albumin promoter and 2 copies of a human prothrombin enhancer is administered before the exogenously produced natural or recombinant α-galactosidase A or a small molecule capable of treating Fabry disease.

22. A method of treating a subject diagnosed as having Pompe disease comprising first administering a gene therapy vector encoding α-glucosidase under the control of a liver specific promoter and optionally, at least one copy of a tissue specific enhancer followed by administration of:

(a) an exogenously produced natural or recombinant α-glucosidase;

(b) a small molecule capable of treating Pompe disease, or

(c) both (a) and (b),

such that the Pompe disease is treated.

23. A composition useful for treating a lysosomal storage disease comprising a gene therapy vector encoding a lysosomal hydrolase under the control of a tissue specific regulatory element and (a) an exogenously produced natural or recombinant lysosomal hydrolase; (b) a small molecule capable of treating a lysosomal storage disease or (c) both (a) and (b).

24. The composition of claim 23, where the gene therapy vector encoding a lysosomal hydrolase encodes α-galactosidase A.

25. The composition of claim 23, where the gene therapy vector encoding a lysosomal hydrolase encodes α-glucosidase.

26. The composition of claim 23, where the gene therapy vector is a viral vector.

27. The composition of claim 26, where the viral vector is chosen from AAV1, AAV2, AAV5, AAV7 and AAV8.

28. The composition of claim 23, where the exogenously produced natural or recombinant lysosomal hydrolase is chosen from α-galactosidase A and α-glucosidase.

29. The composition of claim 23, where the tissue specific regulatory element is a liver specific promoter.

30. The composition of claim 29, where the liver specific promoter is an albumin promoter.

31. The composition of claim 23, where the tissue specific regulatory element is a tissue specific enhancer.

32. The composition of claim 31, where the tissue specific enhancer is a human prothrombin enhancer.

33. The composition of claim 23, where the small molecule capable of treating a lysosomal storage disease is chosen from deoxynojirimycin, N-propyldeoxynojirimycin, N-butyldeoxynojirimycin, N-butyldeoxygalactonojirimycin, N-pentlydeoxynojirimycin, N-heptyldeoxynojirimycin, N-pentanoyldeoxynojirimycin, N-(5-adamantane-1-ylmethoxy)pentyl)-deoxynojirimycin, N-(5-cholesteroxypentyl)-deoxynojirimycin, N-(4-adamantanemethanylcarboxy-1-oxo)-deoxynojirimycin, N-(4-adamantanylcarboxy-1-oxo)-deoxynojirimycin, N-(4-phenantrylcarboxy-1-oxo)-deoxynojirimycin, N-(4-cholesterylcarboxy-1-oxo)-deoxynojirimycin, or N-(4-b-cholestanylcarboxy-1-oxo)-deoxynojirimycin, D-threo-1-phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (P4), D-threo-4'-hydroxy-1-phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (4'-hydroxy-P4), D-threo-1-(3',4'-trimethylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (trimethylenedioxy-P4), D-threo-1-(3',4'-methylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (methylenedioxy-P4) and D-threo-1-(3',4'-ethylenedioxy)phenyl-2-palmitoylamino-3-pyrrolidino-1-propanol (ethylenedioxy-P4 or D-t-et-P4).

34. A composition useful for treating Fabry disease comprising a gene therapy vector encoding α-galactosidase A under the control of a human albumin promoter and 2 copies of a human prothrombin enhancer and:

(a) an exogenously produced natural or recombinant α-galactosidase A;

(b) a small molecule capable of treating Fabry disease, or

(c) both (a) and (b).

35. A composition useful for treating Pompe disease comprising a gene therapy vector encoding α-glucosidase under the control of a liver specific promoter and optionally at least one tissue specific enhancer and:

a) an exogenously produced natural or recombinant α-glucosidase;

b) a small molecule capable of treating Pompe disease or

(c) both (a) and (b).